Metabolic changes during the transition from the fed to the acute feed-deprived state in prepuberal and mature gilts

C. R. Barb, R. R. Kraeling, G. B. Rampacek and C. R. Dove

The online version of this article, along with updated information and services, is located on the World Wide Web at:

http://jas.fass.org

American Society of Animal Science

www.asas.org

Metabolic Changes During the Transition from the Fed to the Acute Feed-Deprived State in Prepuberal and Mature Gilts[1]

C. R. Barb*,[2], R. R. Kraeling*, G. B. Rampacek[†], and C. R. Dove[‡]

*ARS, USDA, Athens, GA 30613; [†]Department of Animal and Dairy Science, University of Georgia, Athens 30602; and [‡]Department of Animal and Dairy Science, University of Georgia, Tifton 31793

ABSTRACT: The present study compared the metabolic response to acute feed deprivation in ovariectomized prepuberal (P; n = 6), 62 ± 2 kg BW, and mature (M; n = 6) gilts, 124 ± 4 kg BW. Blood was collected at 1, 2, 3, 4, 6, 8, 12, 16, 20, 24, 28, 32, 40, and 48 h after initiation of feeding. Samples were quantified for glucose, insulin, free fatty acids (FFA), β-hydroxybutyrate (HBA), IGF-I, IGF binding proteins (BP)-1 to -4, cortisol, and amino acids (AA). At 24 h, blood samples were collected every 15 min for 8 h and an additional 1 h after i.v. injection of GnRH (.2 μg/kg BW) and growth hormone-releasing factor (GRF; 1 μg/kg BW). Samples were assayed for growth hormone (GH) and LH. Serum insulin concentrations were lower ($P < .05$) in P gilts. Plasma glucose concentrations were similar. Serum FFA concentrations were greater ($P < .05$) in P gilts. Serum concentrations of HBA were greater ($P < .001$) in P gilts at 48 h. Serum IGF-I concentrations were lower ($P < .05$) in P gilts by 16 h. Serum IGFBP-1-4 levels were similar. Serum cortisol concentrations were similar. Serum concentrations of the essential AA, isoleucine, lysine, threonine, valine, and phenylalanine were greater ($P < .05$) in P gilts at 40 h. Serum LH concentrations and response to GnRH were similar. Basal serum GH concentrations and peak response to GRF were greater ($P < .05$) in P than in M gilts. The transition from fed to unfed state occurs more rapidly in P than in M gilts.

Key Words: Metabolites, Food Deprivation, Gilts, LH, GH

Introduction

Onset of puberty may be linked to attainment of a critical body weight or a minimum percentage of body fat (Frisch, 1984). Alternatively, metabolic mass and food intake or its correlated metabolic rate may be the triggering mechanism (Frisch, 1984). Cameron et al. (1985) reported that the transition from fed to fasted state occurred more rapidly in juvenile than in mature monkeys. Different temporal responses of metabolic hormones and substrates between adults and juveniles were probably related to a greater glucose production rate, higher metabolic rate, smaller energy reserves, and greater growth requirements of juveniles. Cameron et al. (1985) suggested that the dynamic fluctuations of plasma hormones and metabolic substrates that occurred during the postprandial and postabsorptive periods provide signals to the brain that link metabolic status to the activation of the reproductive system. In pigs, nutritional and metabolic perturbations influence endocrine function (Armstrong and Britt, 1987; Cox et al., 1987; Barb et al., 1991b). Therefore, the objective of this study was to identify specific metabolites and metabolic hormones that could potentially serve as signals linking the body's energy regulating system and activation of the reproductive axis. The metabolic response to acute feed deprivation in prepubertal (**P**) and mature (**M**) gilts was compared, because P gilts have smaller energy reserves and greater growth requirements than M gilts.

BARB ET AL.

Materials and Methods

General

Six P crossbred gilts, averaging 130 d of age and 62 ± 2 kg BW, and six crossbred M gilts that had displayed one or more estrous cycles of 18 to 22 d and averaging 240 d of age and 124 ± 4 kg BW were used. All gilts were ovariectomized (**OVX**) 2 wk before the start of the study. Animals were treated humanely, and experimental procedures had prior approval by the USDA-ARS Animal Care Committee. Gilts were fed to appetite twice daily at 0800 and 1700 over a 2-wk period before the start of the study. Pigs were fed a corn-soybean meal diet (14% crude protein) supplemented with vitamins and minerals, according to National Research Council (1988) guidelines. Pigs were exposed to a constant ambient temperature of 22°C and an artificial 12:12 h light:dark photoperiod in individual pens in an environmentally controlled building. Each gilt was fitted nonsurgically with an indwelling jugular vein catheter the day before blood sampling (Barb et al., 1982). Before blood sampling began, backfat thickness (**BF**) was assessed using ultrasound (Renco Lean Meter, Renco Corp., Minneapolis, MN). Pigs were fed at 0800 (time 0), feed was removed at 0900, and pigs remained without feed for 48 h. Blood was collected at 1, 2, 3, 4, 6, 8, 12, 16, 20, 24, 28, 32, 40, and 48 h after initiation of feeding. Selected samples were assayed for glucose, insulin, FFA, β-hydroxybutyrate (**HBA**), IGF-I, IGF-I binding proteins (**IGFBP**), cortisol, and amino acids (**AA**). At 24 h, blood samples were collected every 15 min for 8 h and an additional 1 h after i.v. injection of GnRH (.2 μg/kg BW) and GH-releasing factor (**GRF**; 1 μg/kg BW). Blood samples were allowed to clot at 4°C for 24 h and serum was harvested, except for samples for glucose and AA, which were drawn into heparinized tubes containing sodium fluoride. Samples were stored at –20°C until they were assayed for GH and LH by RIA.

Metabolite and Hormone Assays. Serum concentrations of GH (Barb et al., 1991a), LH (Kesner et al., 1987), IGF-I (Whitley et al., 1995), and cortisol (Barb et al., 1992) were quantified by RIA as described previously. Sensitivities of the assays were .4 ng/mL, .15 ng/mL, .01 ng/mL, and 1 ng/mL for GH, LH, IGF-I, and cortisol, respectively. Intra- and interassay CV were 3.2 and 13.6% for GH, 4.8 and 9.0% for LH, 3.2 and 3.5% for IGF-I, and 1.4 and 4.4% for cortisol, respectively.

Serum insulin concentrations were measured with an RIA kit (ICN Biomedical, Costa Mesa, CA). Samples were assayed for glucose using a glucose oxidase kit (Sigma Chemical, St. Louis, MO), FFA using a colorimetric procedure (Itaya and Ui, 1965), and HBA with an oxidase kit (Sigma). Plasma AA were determined by treating samples with 10% sulfosalicylic acid solution to precipitate proteins and

centrifuged $(10,000 \times g)$ before analysis. The resulting supernatant was used for determination of amino acids by ion exchange chromatography (Beckman 6300 Amino Acid Analyzer, Beckman Instruments, Palo Alto, CA).

Serum IGFBP Determination. Serum IGFBP-1, 29,000 M_r; IGFBP-2, 34,000 M_r; IGFBP-3, 39,000 to 43,000 M_r; and IGFBP-4, 24,000 M_r were determined by ligand blotting (Latimer et al., 1993). Briefly, 20 μL of serum was mixed with 6.66 μL of sample buffer, heated for 3 min at 100°C, and loaded onto 12.5% discontinuous nonreducing SDS-polyacrylamide gels. The samples were electrophoresed at 35 mA/gel for 4 to 5 h. Separated proteins were transferred to PVDF membrane (BioRad, Hercules, CA), and the blots were incubated with 2×10^6 cpm [^{125}I]IGF-I /mL. Insulin-like growth factor-BP were visualized by autoradiography (Kodak X-AR film, Eastman Kodak Co., Rochester NY; –80°C for 12 d), sized by comparison to prestained protein standards, and quantified by computer-assisted image analysis with use of a Dage CCD-72 camera (Dage-MTI, Michigan City, IN) and IM-3000 software (Analytical Imaging Concepts, Irvine, CA). The identity of IGFBP-1 in porcine sera has been confirmed using specific immunoblot analysis (Latimer et al., 1993). Identity of IGFBP-2 was also confirmed using specific immunoblot analysis (ProtoBlot Western blot, Promega, Madison, WI; McCusker et al., 1990). Identities of IGFBP-3 and IGFBP-4 in porcine sera have been determined by N-terminal sequence determination (Coleman et al., 1991).

Statistical Analysis. Differences in serum metabolite and hormone concentrations across time and between P and M gilts were determined by subjecting data to a split-plot-in-time analysis of variance using the GLM procedures of SAS (1988). Age, pig, and time were discrete (class) variables. Age and time and the age × time interaction were tested with pig-within-age and time × pig-within-age as the error terms, respectively. Differences between ages within a time were determined by least squares contrasts (SAS, 1988). For each gilt, mean serum hormone concentrations, basal hormone concentrations, the number of hormone pulses, and hormone pulse amplitude were determined by Pulsar analysis, using a 1% criterion of variation (Merriam and Wachter, 1982), during the 8-h sampling period starting at 24 h after feeding. Data were then subjected to one-way ANOVA. Each of the serum IGFBP-1 to -4 was expressed as a percentage of the total IGFBP units for each pig. Data were then subjected to a split-plot-in-time analysis of variance using the GLM procedures of SAS (1988) as described above. Difference in BF between P and M gilts was determined by subjecting data to analysis of variance using the general linear model procedure of SAS (1988).
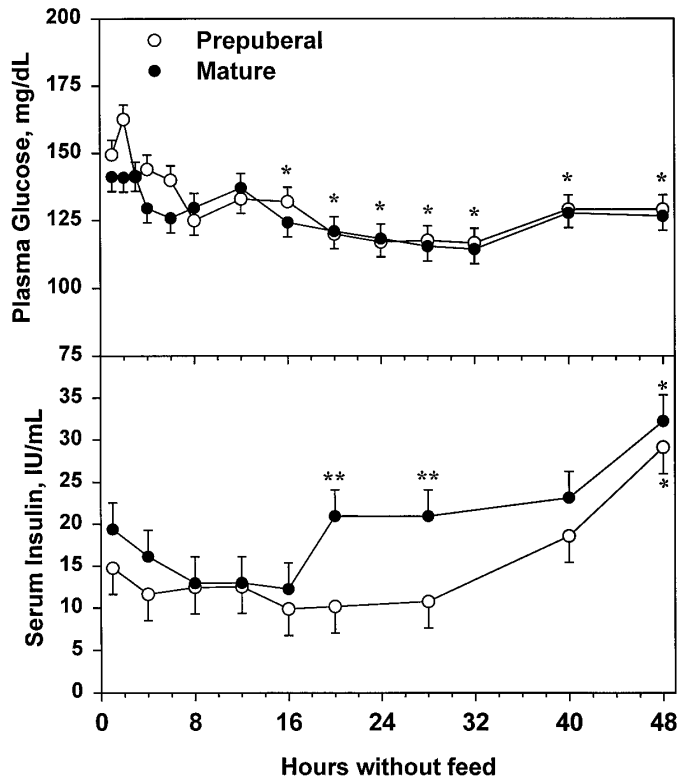
Figure 1. Mean (± SE) plasma glucose and serum insulin concentrations for prepuberal (n = 6) and mature (n = 6) gilts for 48 h without feed. *Differ from h 1 ($P <$ .05), **prepuberal different from mature gilts ($P <$ .05).

Results

Metabolites and Metabolic Hormones. Backfat thickness was greater ($P <$ .001) in M (22 ± 1 mm) than in P (14 ± 1 mm) gilts. Plasma glucose concentrations were similar in P and M gilts and decreased ($P <$ .05) in both groups by 16 h and then remained unchanged (Figure 1). Serum insulin concentrations were similar in P and M gilts during the initial 16 h of feed deprivation but were lower ($P <$ .05) in P than in M gilts at 20 and 28 h of fasting (Figure 1). Serum insulin concentrations increased ($P <$ .05) at 48 h in P and M gilts compared with h 1 (Figure 1). Serum FFA concentrations increased ($P <$ .05) by 12 h in P and 20 h in M gilts compared with h 1. Serum FFA levels were greater ($P <$ .05) in P than in M gilts by 12 h and remained greater than in M gilts until 48 h (Figure 2). Serum HBA concentrations increased ($P <$ .05) by 32 h in P and 40 h in M gilts compared with h 1. Serum concentrations of HBA were greater ($P <$ .001) in P than in M gilts at 48 h of feed deprivation. Serum IGF-I concentrations decreased ($P <$ .05) by 8 h in P gilts and 28 h in M gilts compared with h 1. Serum IGF-I concentrations were lower ($P <$ .05) in P than in M gilts by 16 h and remained lower through 48 h (Figure 3). Percentage of the total IGFBP that were IGFBP-1, -2, and -4 were similar for P and M

animals and remained unchanged during feed deprivation. The IGFBP-3 made up the greatest ($P <$ .05) percentage of the total BP, was similar for P and M animals, and remained unchanged in both age groups during the period without feed (Figure 4). Serum cortisol concentrations were similar during feed deprivation and averaged 46 ± 3 and 42 ± 3 ng/mL for P and M gilts, respectively.

Amino Acids. An age × time interaction ($P <$ .05) was detected for isoleucine, lysine, threonine, and valine. In addition, there was a significant ($P <$ .04) age effect for phenylalanine. Plasma concentrations of these AA were greater ($P <$ .05) in P than in M gilts by 40 h without feed (Figure 5). The ratio of tryptophan to other large neutral AA (**LNAA**) in the serum was greater ($P <$ .05) in M than in P gilts and remained unchanged during feed deprivation (Figure 6a). The ratio of tyrosine to other LNAA did not change during the first 8 h without feed, then it decreased ($P <$ .05) from 12 to 20 h and was greater ($P <$ .005) in M than in P animals during this period. However, by 24 h without feed these ratios were similar (Figure 6b). Throughout the period without feed, plasma concentrations of alanine, aspartate, glutamate, glutamine, glycine, and hydroxy-L-proline were greater ($P <$ .05) in P than in M gilts (Table 1).

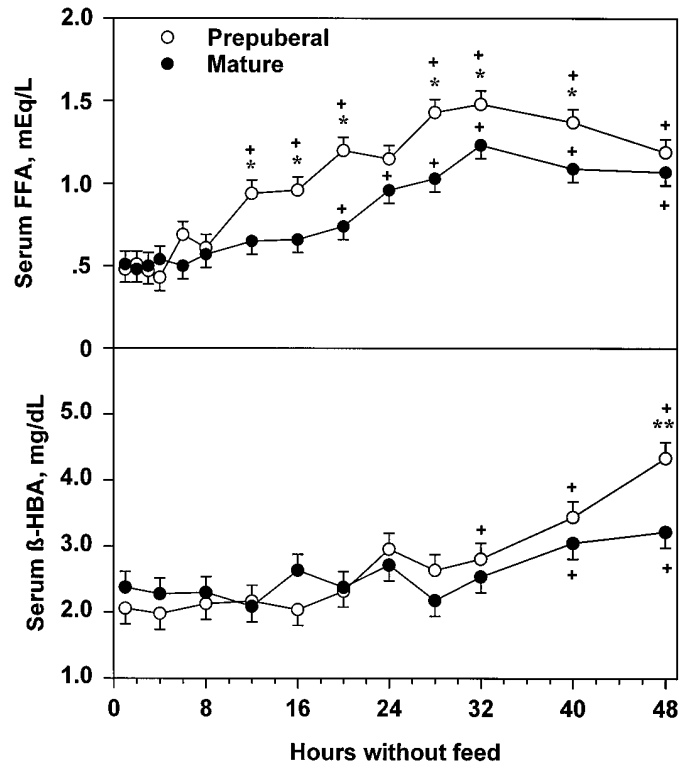Figure 2. Mean (± SE) serum free fatty acids (FFA) and β-hydroxybutyrate (HBA) concentrations for prepuberal (n = 6) and mature (n = 6) gilts for 48 h without feed. †Differ from h 1 ($P <$ .05), prepuberal different from mature gilts, *$P <$ .05, **$P <$ .001.

Luteinizing Hormone and Growth Hormone. Serum LH concentrations and LH response to GnRH were similar for P and M gilts (Figure 7). Mean and basal serum LH concentrations, LH pulse frequency, and LH pulse amplitude as determined by Pulsar analysis were similar for P and M gilts (Table 2). An age × time interaction ($P = .076$) was detected for serum GH concentrations (Figure 8). Serum GH concentrations and peak GH response to GRF were greater ($P < .05$) in P than in M gilts (Figure 8). Mean serum GH concentrations, GH pulse frequency, and GH pulse amplitude were similar, but mean basal GH concentration was greater ($P = .05$) in P than in M animals (Table 3).

Discussion

Our results indicate that the response to feed deprivation resulted in a more rapid increase in serum concentrations of FFA and HBA as well as a more precipitous decrease in serum IGF-I concentrations in P compared with M animals. Plasma glucose concentrations decreased by 16 h without feed but were similar between P and M gilts. Serum insulin concentrations, however, were greater in M than in P gilts at 20 and 28 h without feed and increased by 48 h in P and M gilts. Ketone bodies and FFA may directly stimulate the pancreas to release insulin as part of a feedback mechanism for controlling FFA release from adipose tissue during periods without feed (Mayes, 1993). This may in part explain the
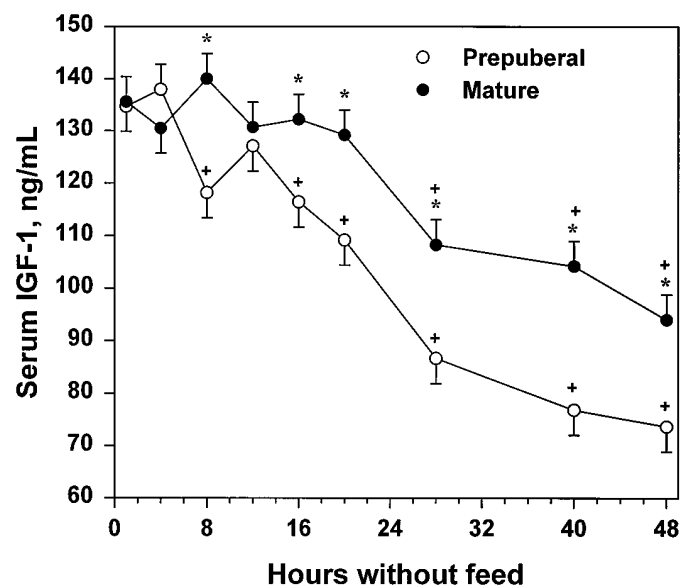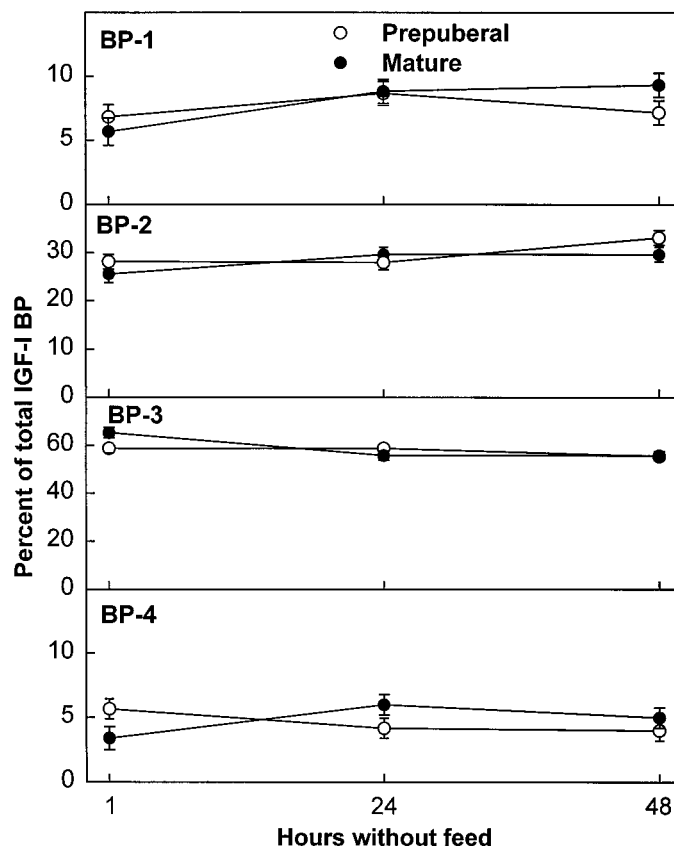

Figure 4. Serum insulin-like growth factor I (IGF) binding protein (IGFBP)-1 to -4 for prepuberal (n = 6) and mature (n = 6) gilts for 48 h without feed. Serum IGFBP-1 to -4 expressed as a percentage of the total IGFBP units (mean ± SE).

increase in insulin observed in the present study. Similarly, in humans (Haymond et al., 1982; Kerr et al., 1983) and in nonhuman primates (Cameron et al., 1985), the rate of adaptation to fast occurred more quickly in juveniles than in adults. Plasma HBA levels increased more rapidly in juveniles than in adults (Haymond et al., 1982; Kerr et al., 1983). In addition, postprandial plasma concentrations of insulin were greater in adults than in juveniles (Kerr et al., 1983). As in pigs, plasma glucose concentrations decreased similarly but unlike in pigs were lower by 44 h of fast in the juvenile compared to the adult primate (Cameron et al., 1985). In the human, with no additional glucose input, liver glycogen stores are gradually depleted between 18 and 24 h of fast. This results in an increase in gluconeogenesis and ketogenesis (Gray et al., 1985). In the present study, a similar sequence of events occurred in both P and M animals. However, in the P gilt these events occurred at a faster rate; serum FFA and HBA concentrations increased more rapidly in P gilts than in M gilts. Thus, the ability of the P gilt to maintain euglycemia during an acute fast may primarily be due to rapid mobilization of alternative energy stores such as FFA.

Figure 3. Mean (± SE) serum insulin-like growth factor I (IGF-I) concentrations for prepuberal) (n = 6) and mature (n = 6) gilts for 48 h without feed. †Differ from h 1 ($P < .05$). *Prepuberal different from mature gilts ($P < .05$).

Metabolic response to feed deprivation may proceed more rapidly in P gilts than in M gilts as a result of an increased rate of energy consumption and a decreased availability of energy substrates. We suggest P gilts have a higher glucose flux rate than M gilts. In support of this idea, the rapid onset of FFA mobilization from peripheral fat depots and maintenance of euglycemia suggests increased hydrolysis of triglycerides and FFA oxidation resulting in a glucose sparing effect.

In human and nonhuman primates, plasma concentrations of AA, such as valine, leucine, isoleucine, alanine, and glutamine, decreased more rapidly in juveniles than in adults in response to an acute fast
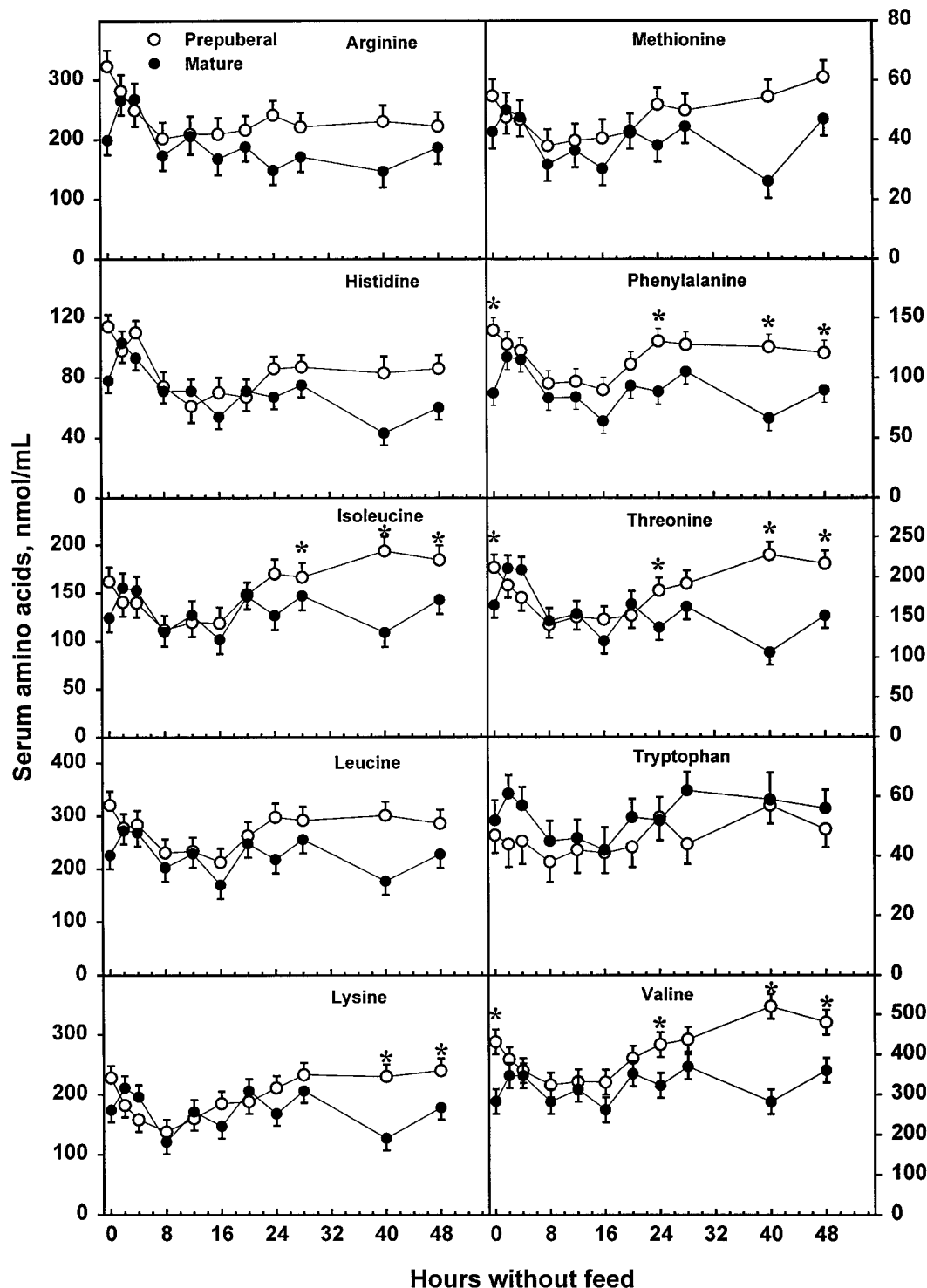

Figure 5. Mean (± SE) serum concentrations of essential amino acids for prepuberal (n = 6) and mature (n = 6) gilts for 48 h without feed. *Prepuberal different from mature gilts ($P < .05$).

Table 1. Mean serum concentrations (nmol/mL) of nonessential amino acids,
ammonia, and urea for prepuberal (n = 6) and mature
(n = 6) gilts for 48 h without feed[a]

Variable	Prepuberal	Mature	SEM	P-value
Alanine	512	311	47	.02
Ammonia	317	193	43	NS[b]
L-Aspartate	62	39	7	.04
Citrulline	51	50	6	NS
Cystine	13	12	2	NS
L-Glutamate	342	196	40	.03
L-Glutamine	379	244	28	.01
Glycine	982	736	78	.05
Hydroxy-L-proline	64	42	5	.02
Ornithine	119	112	12	NS
Phosphoserine	23	20	1	NS
Proline	297	231	30	NS
Serine	185	137	16	NS
Taurine	287	219	32	NS
Tyrosine	88	74	10	NS
Urea	4,548	4,328	330	NS

[a]Data expressed as mean of samples collected at h 1, 12, 24, and 48.
[b]NS = not significant.

(Haymond et al., 1982; Cameron et al., 1985). This would suggest that AA were being mobilized from peripheral protein stores to provide substrates for gluconeogenesis in juveniles. In the present study there was no discernible pattern in serum concentrations of AA during the period without feed. Serum concentrations of essential AA were either similar between P and M gilts or greater by 40 h without feed in P gilts. Moreover, it is doubtful that AA were being deaminated for gluconeogenesis, because serum concentrations of urea and ammonia were unchanged during the period without feed in P and M animals. Pigs possesses large stores of adipose tissue (Allen et al., 1976) that would provide an adequate alternative energy source. Thus, euglycemia was probably maintained by mobilizing lipid stores as an alternative energy source instead of AA.

The nutritional status of an animal influences serum IGF-I concentrations. Fasting or feeding a diet deficient in energy or protein was associated with a

Table 2. Mean and basal serum LH concentrations, LH pulse frequency, and LH
pulse amplitude for prepuberal and mature gilts at 24 to 32 h without feed[a]

Age	No. of gilts	Mean LH concentrations, ng/mL	Basal LH concentrations, ng/mL	LH pulse frequency, pulses/8 h	LH pulse[b] amplitude, ng/mL
Prepuberal	6	1.2 ± .2	1.0 ± .2	7.2 ± .4	.7 ± .1
Mature	6	1.4 ± .2	1.2 ± .1	7.8 ± .5	.7 ± .1

[a]Values expresses as mean ± SE.
[b]Amplitude = pulse height − basal concentration.

Table 3. Mean and basal serum GH concentrations, GH pulse frequency, and GH
pulse amplitude for prepuberal and mature gilts at 24 to 32 h without feed[a]

Age	No. of gilts	Mean GH concentrations, ng/mL	Basal GH concentrations, ng/mL	GH pulse frequency pulses/8 h	GH pulse[b] amplitude ng/mL
Prepuberal	6	2.1 ± .4	1.8 ± .3[c]	2.7 ± .3	2.2 ± .5
Mature	6	1.5 ± .1	1.0 ± .1[d]	2.0 ± .3	3.0 ± .5

[a]Values expressed as mean ± SE.
[b]Amplitude = pulse height − basal concentration.
[c,d]Mean in a column with different superscripts are different ($P = .05$).

reduction in serum IGF-I levels in humans (Smith et al., 1995), beef heifers (Armstrong et al., 1993), and rats (Emler and Schalch, 1987). Results from the present study are consistent with the response to feed deprivation in barrows (Buonomo and Baile, 1991), prepuberal gilts (Charlton et al., 1993), and neonatal pigs (Campion et al., 1986). The mechanisms responsible for reduction in serum IGF-I concentrations during periods without feed or malnutrition are not clear. Nutritional restriction is associated with a decrease in number of hepatic GH receptors (Breier et al., 1988; Maiter et al., 1988) and GH receptor binding capacity (Maes et al., 1983). Moreover, dietary protein restriction or fast suppressed IGF-I response to GH treatment (Phillips and Unterman, 1984; Thissen et al., 1990), indicating GH resistance by a postreceptor mechanism (Clemmons and Underwood, 1991). In addition, fasting reduced hepatic (Emler and Schalch, 1987) and skeletal muscle (VandeHaar et al., 1991) IGF-I mRNA concentrations but failed to alter the half-life of IGF-I in blood (Hodgkinson et al., 1987). Because circulating concen-

Figure 7. Mean (± SE) serum LH concentrations and LH response to GnRH for prepuberal (n = 6) and mature (n = 6) gilts at 24 to 33 h without feed.

trations of IGF-I are dependent on IGFBP, the marked decrease in serum IGF-I concentrations in the present study in response to feed deprivation may reflect changes in IGFBP concentrations. However, we failed to observe an acute effect on IGFBP in P and M gilts. This is in contrast to reports on humans (Cohick and Clemmons, 1993) and neonatal pigs (McCusker et al., 1991), whereas others have failed to demonstrate an effect of nutrient restriction on IGFBP in lactating cows (McGuire et al., 1995) and beef heifers (Armstrong et al., 1993). In castrated sheep, feed deprivation decreased IGFBP-3 and increased IGFBP-2, but feed deprivation in pregnant ewes decreased IGFBP-3 without affecting IGFBP-2 (Gallaher et al., 1992).
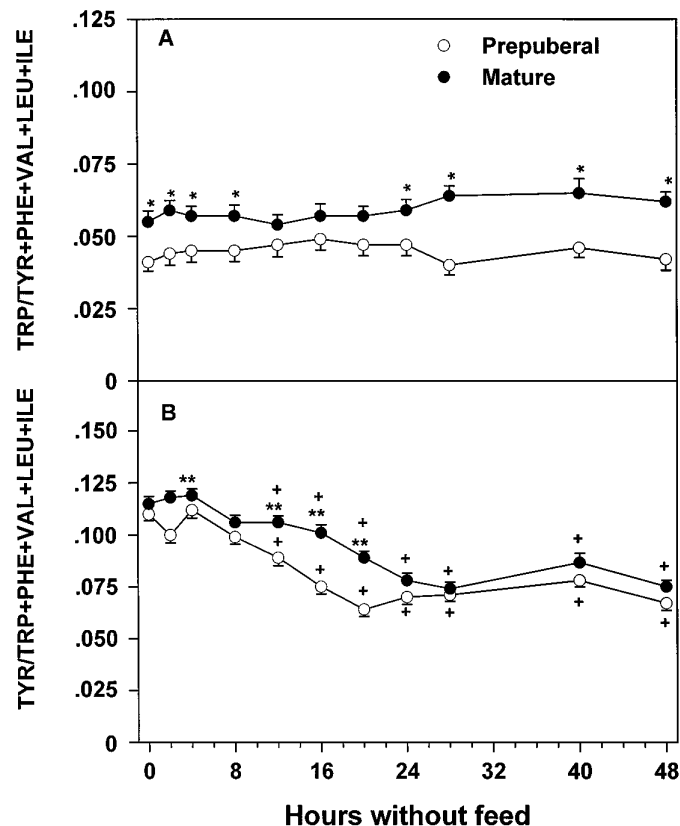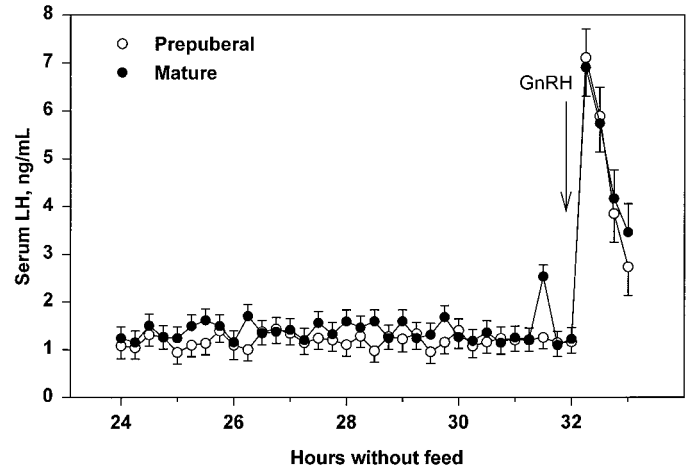

Figure 6. Serum ratios of tyrosine (TYR)/tryptophan (TRP) + phenylalanine (PHE) + valine (VAL) + leucine (LEU) + isoleucine (ILE; panel A) and TRP/TYR + PHE + VAL + LEU + ILE (panel B) in prepuberal (n = 6) and mature (n = 6) gilts for 48 h without feed. +Differ from h 1 (P < .05), prepuberal different from mature gilts, *P < .05, **P < .005.
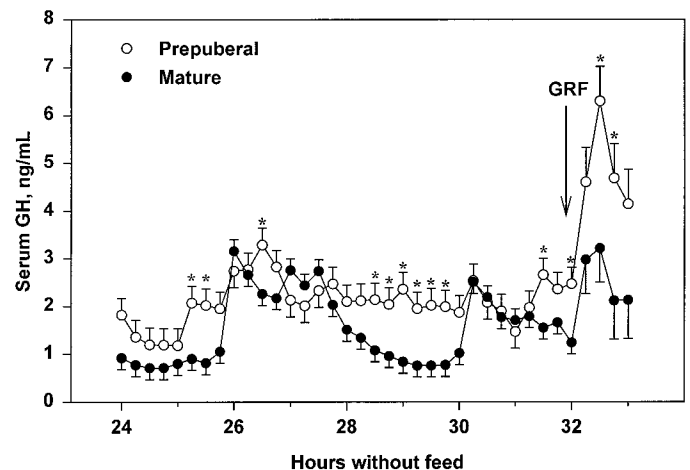

Figure 8. Mean (± SE) serum growth hormone (GH) concentrations and GH response to GH releasing factor (GRF) for prepuberal (n = 6) and mature (n = 6) gilts at 24 to 33 h without feed. An age × time interaction (P = .075) was detected. *Prepuberal different from mature gilts (P < .05).

These differences may in part be related to species, type of fast (acute vs nutrient restriction), or physiological state.

Alteration in AA profiles during periods of fast or feed restriction have been proposed as a mechanism to explain alterations in GnRH secretion (Steiner et al., 1983). Changes in LNAA ratios affect their transport into the brain (Fernstrom, 1983). Consequently, their availability as precursors for monoamine neurotransmitter synthesis may change during nutrient deprivation. Indolamine and catecholamine pathways affect LH (Kraeling and Barb, 1990) and GH (Buonomo and Baile, 1990) secretion in pigs. Therefore, changes in peripheral LNAA ratios might ultimately influence GnRH and GRF/somatostatin secretion. In the present study the ratio of tryptophan to other LNAA remained unchanged during feed deprivation, but the ratio of tyrosine to other LNAA decreased faster in P than in M animals. The ratio of each LNAA to the sum of all LNAA is a good predictor of brain LNAA concentrations (Fernstrom, 1983). It is conceivable that brain neurotransmitter concentrations were altered in the present study. This may explain why GH concentrations were greater in P gilts than in M gilts. Alternatively, elevated serum GH concentrations and increased response to GRF observed in the P gilts may be age-related (Dubreuil et al., 1987).

Booth (1990) demonstrated that administering glucose to feed-restricted gilts and the associated increase in plasma insulin concentrations induced a rapid increase in episodic LH secretion similar to that observed in response to realimentation. In the present study, the ability of the P and M gilts to maintain euglycemia may explain why LH secretion and response to GnRH were not different.

Contemporary models of energy regulation emphasize physiological signals that control energy intake, partitioning and expenditure and their detectors. It is hypothesized that mechanisms regulating energy balance are sensitive to metabolic signals generated by changes in the oxidation of metabolic fuels and could account for positive correlations between fat, fertility, and endocrine function (Wade and Schneider, 1992).

Changes in fat metabolism, serum ketone body, and IGF-I concentrations during periods of fast or nutrient deprivation could influence activity of hypothalamic neurons (Oomura et al., 1975; Abe et al., 1983; Hiney et al., 1991) and(or) pituitary responsiveness to GnRH and GRF (Quabbe et al., 1983, 1991; Barb et al., 1991b, 1995; Whitley et al., 1995). Results from the present study do not allow identification of specific candidates for a role in signaling metabolic status to the neuroendocrine axis that regulates LH and GH secretion. However, we suggest the FFA and ketone bodies along with IGF-I may serve as possible metabolic signals linking the body's energy regulating system with reproduction and growth processes. Fu-

ture studies are needed to address the role of specific metabolites and metabolic hormones in modulating LH and GH secretion in pigs.

Implications

Results from the present study do not allow identification of specific metabolites for a role in signaling metabolic status to the neuroendocrine axis. However, they do provide potential candidates for future studies designed to investigate the role these signals play in modulating the endocrine system in the pig.

Literature Cited

Abe, H., M. E. Molitch, J. J. Van Wyk, and L. E. Underwood. 1983. Human growth hormone and somatomedin C suppress the spontaneous release of growth hormone in unanesthetized rats. Endocrinology 113:1319.

Allen, C. E., D. C. Bietz, D. A. Cramer, and R. G. Kauffman. 1976. Biology of Fat in Meat Animals. North Central Reg. Res. Publ. No. 234:1.

Armstrong, J. D., and J. H. Britt. 1987. Nutritionally-induced anestrus in gilts: Metabolic and endocrine changes associated with cessation and resumption of estrous cycles. J. Anim. Sci. 65: 508.

Armstrong, J. D., W. S. Cohick, R. W. Harvey, E. P. Heimer, and R. M. Campbell. 1993. Effect of feed restriction on serum somatotropin, insulin-like growth factor-I-(IGF-I) and IGF binding proteins in cyclic heifers actively immunized against growth hormone releasing factor. Domest. Anim. Endocrinol. 10:315.

Barb, C. R., G. M. Derochers, B. Johnson, R. V. Utley, W. J. Chang, G. B. Rampacek, and R. R. Kraeling. 1992. N-methyl-d,l-aspartate stimulates growth hormone and prolactin but inhib its luteinizing hormone secretion in the pig. Domest. Anim. Endocrinol. 9:225.

Barb, C. R., M. J. Estienne, R. R. Kraeling, D. N. Marple, G. B. Rampacek, C. H. Rahe, and J. L. Sartin. 1991a. Endocrine changes in sows exposed to elevated ambient temperature during lactation. Domest. Anim. Endocrinol. 8:117.

Barb, C. R., R. R. Kraeling, J. B. Barrett, G. B. Rampacek, R. M. Campbell, and T. F. Mowles. 1991b. Serum glucose and free fatty acids modulate growth hormone and luteinizing hormone secretion in the pig. Proc. Soc. Exp. Biol. Med. 198:636.

Barb, C. R., R. R. Kraeling, and G. B. Rampacek. 1995. Glucose and free fatty acid modulation of growth hormone and luteinizing hormone secretion by cultured porcine pituitary cells. J. Anim. Sci. 73:1416.

Barb, C. R., R. R. Kraeling, G. B. Rampacek, E. S. Fonda, and T. E. Kiser. 1982. Inhibition of ovulation and LH secretion in the gilt after treatment with ACTH or hydrocortisone. J. Reprod. Fertil. 64:85.

Booth, P. J. 1990. Metabolic influences on hypothalamic-pituitary ovarian function in the pig. J. Reprod. Fertil. (Suppl. 40):89.

Breier, B. H., P. D. Gluckman, and J. J. Bass. 1988. Influence of nutritional status and oestradiol-17 beta on plasma growth hormone, insulin-like growth factor-I and -II and the response to exogenous growth hormone in young steers. J. Endocrinol. 118:243.

Buonomo, F. C., and C. A. Baile. 1990. The neurophysiological regulation of growth hormone secretion. Domest. Anim. Endocrinol. 7:435.

Buonomo, F. C., and C. A. Baile. 1991. Influence of nutritional deprivation on insulin-like growth factor-I, somatotropin and metabolic hormones in swine. J. Anim. Sci. 69:755.

Cameron, J. L., D. J. Koerker, and R. A. Steiner. 1985. Metabolic changes during maturation of male monkeys: Possible sign for onset of puberty. Am. J. Physiol. 249:E385.

Campion, D. R., R. H. McCusker, F. C. Buonomo, and W. K. Jones,Jr. 1986. Effect of fasting neonatal piglets on blood hormone and metabolite profiles and on skeletal muscle metabolism. J. Anim. Sci. 63:1418.

Charlton, S. T., J. R. Cosgrove, D. R. Glimm, and G. R. Foxcroft. 1993. Ovarian and hepatic insulin-like growth factor-I gene expression and associated metabolic responses in prepubertal gilts subjected to feed restriction and refeeding. J. Endocrinol. 139:143.

Clemmons, D. R., and L. E. Underwood. 1991. Nutritional regulation of IGF-I and IGF binding proteins. Annu. Rev. Nutr. 11:393.

Cohick, W. S., and D. R. Clemmons. 1993. The insulin-like growth factors. Annu. Rev. Physiol. 55:131.

Coleman, M. E., Y.-C. E. Pan, and T. D. Etherton. 1991. Identification and NH2 terminal amino acid sequence of three insulin-like growth factor-binding proteins in procine serum. Biochem. Biophys. Res. Commun. 181:1131.

Cox, N. M., M. J. Stuart, T. G. Althen, W. A. Bennett, and H. W. Miller. 1987. Enhancement of ovulation rate in gilts by increasing dietary energy and administering insulin during follicular growth. J. Anim. Sci. 64:507.

Dubreuil, P., G. Pelletier, D. Petitclerc, H. Lapierre, Y.Couture, P. Brazeau, P. Gaudreau, and J. Morisset. 1987. Influence of age and sex on basal secretion of growth hormone (GH) and on GH-induced release by porcine GH-releasing factor pGRF(1-29NH2) in growing pigs. Domest. Anim. Endocrinol. 4:299.

Emler, C. A., and D. S. Schalch. 1987. Nutritionally induced changes in hepatic insulin-like growth factor (IGF-I) gene expression in rats. Endocrinology 120:832.

Fernstrom, J. D. 1983. Role of precursor availability in control of monoamine biosynthesis in brain. Physiol. Rev. 63:484.

Frisch, R. E. 1984. Body fat, puberty and fertility. Biol. Rev. 59:161.

Gallaher, B. W., B. H. Breier, M. H. Oliver, J. E. Harding, and P. D. Gluckman. 1992. Ontogenic differences in the nutritional regulation of circulating IGF binding proteins in sheep plasma. Acta Endocrinol. 18:49.

Gray, C. G., R. W. Phillips, and M. E. Tumbleson. Swine in biomedical research. 1985. In: M.E. Tumbleson (Ed.) Mechanisms of Bioenergetic Homeostasis During Exercise: A General Model. p 515. Plenum Press, New York.

Haymond, M. W., I. E. Karl, W. L. Clarke, A. S. Pagliara, and J. V. Santiago. 1982. Differences in circulating gluconeogenic substrates during short-term fasting in men, women, and children. Metabolism 31:33.

Hiney, J. K., S. R. Ojeda, and W. L. Dees. 1991. Insulin-like growth factor-I: A possible metabolic signal involved in the regulation of female puberty. Neuroendocrinology 54:420.

Hodgkinson, S. C., S. R. Davis, B. D. Burleigh, H. V. Henderson, and P. D. Gluckman. 1987. Metabolic clearance rate of insulin-like growth factor-I in fed and starved sheep. J. Endocrinol. 115:233.

Itaya, K., and M. Ui. 1965. Colorimetric determination of free fatty acids in biological fluids. J. Lipid Res. 6:16.

Kerr, D. S., I. L. Hansen, and M. M. Levy. 1983. Metabolic and hormonal responses of children and adolescents to fasting and 2-deoxyglucose. Metabolism 32:951.

Kesner, J. S., R. R. Kraeling, G. B. Rampacek, and B. Johnson. 1987. Absence of an estradiol-induced surge of luteinizing hormone in pigs receiving unvarying pulsatile gonadotropin-releasing hormone stimulation. Endocrinology 121:1862.

Kraeling, R. R., and C. R. Barb. 1990. Hypothalamic control of gonadotrophin and prolactin secretion in pigs. J. Reprod. Fert. (Suppl. 40):3.

Latimer, A. M., G. J. Hausman, R. H. McCusker, and F. C. Buonomo. 1993. The effects of thyroxine on serum and tissue concentrations on insulin-like growth factors (IGF-I and -II) and IGF-binding proteins in the fetal pig. Endocrinology 133:1312.

Maes, M., L. E. Underwood, and J. M. Ketelslegers. 1983. Plasma somatomedin-C in fasted and refed rats: close relationship between changes in liver somatogenic but not lactogenic binding sites. J. Endocrinol. 97:243.

Maiter, D., L. E. Underwood, M. Maes, M. L. Davenport, and J. M. Ketelslegers. 1988. Different effects of intermittent and continuous growth hormone (GH) administration of serum somatomedin-C/insulin-like growth factor-1 and liver GH receptors in hypophysectomized rats. Endocrinology 123:1053.

Mayes, P. A. 1993. Harper's biochemistry. In: R. K. Murray, D. K. Granner, P. A. Mayes, and V. W. Rodwell (Ed.) Integration of Metabolism and the Provision of Tissue Fuels. p 279. Appleton and Lange, Norwalk, CT.

McCusker, R. H., C. Camacho-Hubner, M. L. Bayne, M. A. Cascieri, and D. R. Clemmons. 1990. Insulin-like growth factor (IGF) binding to human fibroblast and glioblastoma cells: The modulating effect of cell released IGF binding proteins (IGFBPs). J. Cell. Physiol. 144:244.

McCusker, R. H., W. S. Cohick, W. H. Busby, and D. R. Clemmons. 1991. Evaluation of the developmental and nutritional changes in porcine insulin-like growth factor-binding protein-1 and -2 serum levels by immunoassay. Endocrinology 129:2631.

McGuire, M. A., D. E. Bauman, D. A. Dwyer, and W. S. Cohick. 1995. Nutritional modulation of the somatotropin/insulin-like growth factor system: Response to feed deprivation in lactating cows. J. Nutr. 125:493.

Merriam, G. R., and K. W. Wachter. 1982. Algorithms for the study of episodic hormone secretion. Am. J. Physiol. 6:E310.

NRC. 1988. Nutrient Requirements of Swine (9th Ed.). National Academy Press, Washington, DC.

Oomura, Y., T. Nakamura, M. Sugimori, and Y. Yamada. 1975. Effect of free fatty acid on the rat lateral hypothalamic neurons. Physiol. Behav. 14:483.

Phillips, L. S., and T. G. Unterman. 1984. Somatomedin acturity in disorders of nutrition and metabolism. J. Clin. Endocrinol. & Metab. 13:145.

Quabbe, H.-J., C. Bumke-Vogt, J. R. Iglesias-Rozas, S. Freitag, and N. Breitinger. 1991. Hypothalamic modulation of growth hormone secretion in the Rhesus monkey: Evidence from intracerebroventricular infusions of glucose, free fatty acid, and ketone bodies. J. Clin. Endocrinol. & Metab. 73:765.

Quabbe, H. J., M. Trompke, and A. S. Luyckx. 1983. Influence of ketone body infusion on plasma growth hormone and glucagon in man. J. Clin. Endocrinol. & Metab. 57:613.

SAS. 1988. SAS User's Guide (Release 6.03). SAS Inst. Inc., Cary, NC.

Smith, W. J., L. E. Underwood, and D. R. Clemmons. 1995. Effects of caloric or protein restriction on insulin-like growth factor-I (IGF-I) and IGF-binding proteins in children and adults. J. Clin. Endocrinol. & Metab. 80:443.

Steiner, R. A., J. L. Cameron, T. H. McNeill, D. K. Clifton, and W. J. Bremmer. 1983. Neuroendocrinological aspects of reproduction. In: R. L. Norman (Ed.) Metabolic Signals for the Onset of Puberty. p 183. Academic Press, New York.

Thissen, J. P., S. Triest, M. Maes, L. E. Underwood, and J. M. Ketelslegers. 1990. The decreased plasma concentration of insulin-like growth factor-I in protein-restricted rats is not due to decreased numbers of growth hormone receptors on isolated hepatocytes. J. Endocrinol. 124:159.

VandeHaar, M. J., B. M. Moats-Staats, M. L. Davenport, J. L. Walker, and J. M. Ketelslegers. 1991. Reduced serum concentrations of insulin-like growth factor-I (IGF-I) in protein-restricted growing rats are accompanied by reduced IGF-I mRNA levels in liver and skeletal muscle. J. Endocrinol. 130:305.

Wade, G. N., and J. E. Schneider. 1992. Metabolic fuels and reproduction in female mammals. Neurosci. Biobehav. Rev. 16:235.

Whitley, N. C., C. R. Barb, R. V. Utley, J. M. Popwell, R. R. Kraeling, and G. B. Rampacek. 1995. Influence of stage of the estrous cycle on insulin-like growth factor-I modulation of luteinizing hormone secretion in the gilt. Biol. Reprod. 53:1359.

Citations

This article has been cited by 3 HighWire-hosted articles:
http://jas.fass.org#otherarticles